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APR 25 2002

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**To:** Examiners Phillip Gambel and Paula K. Hutzell  
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The opinion in support of the decision being entered today was not written  
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Paper No. 59

## UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS  
AND INTERFERENCES

MAILED

Ex parte: PETER S. LINSLEY, WILLIAM BRADY,  
JEFFREY A. LEDBETTER AND NITIN K. DAMLE

MAR 26 2002

Appeal No. 1999-2330  
Application No. 08/219,200

PAT. & T.M. OFFICE  
BOARD OF PATENT APPEALS  
AND INTERFERENCES

## ON BRIEF

Before WILLIAM F. SMITH, MILLS and GRIMES, Administrative Patent Judges.  
MILLS, Administrative Patent Judge.

DECISION ON APPEAL

This is a decision on appeal under 35 U.S.C. § 134 from the examiner's final  
rejection of claims 79-94, which are all of the claims pending in this application.

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Claim 79 is illustrative of the claims on appeal and reads as follows:

79. A method for inhibiting T cell proliferation comprising contacting CD28 positive T cells with a soluble B7 fusion protein so as to bind CD28 on the CD28 positive T cells with the soluble B7 protein and thereby inhibiting T cell proliferation.

The references relied upon by the examiner are:

Kahan, "Immunosuppressive therapy," Current Opinion in Immunology, Vol. 4, pp.553-560 (1992)

Yi-qun et al. (Yi-qun), "Differential requirements for co-stimulatory signals from B7 family members by resting versus recently activated memory T cells towards soluble recall antigens," International Immunology, Vol. 8, No. 1, pp. 37-44 (1996)

Perrin et al. (Perrin), "Opposing effects of CTLA4-Ig and Anti-CD80 (B7-1) plus Anti-CD86 (B7-2) on experimental allergic encephalomyelitis," Journal of Neuroimmunology, Vol. 65, pp. 31-39 (1996)

Blazar et al. (Blazar), "Infusion of Anti-B7.1 (CD80) and Anti-B7.2 (CD86) monoclonal antibodies inhibits murine graft-versus-host disease lethality in part via direct effects on CD4<sup>+</sup> and CD8<sup>+</sup> T cells," Journal of Immunology, Vol. 157, pp. 3250-3259 (1996)

The references relied upon by the appellants are:

Freeman et al. (Freeman), "B7, A new member of the Ig superfamily with unique expression on activated and neoplastic B cells," Journal of Immunology, Vol. 143, No. 8, pp. 2714-2722 (1989)

Lenschow, et al. (Lenschow), "Long-term survival of xenogenic pancreatic islet grafts induced by CTLA4Ig," Science, Vol. 257, pp. 789-792 (1992)

### Background

The claimed invention relates to a method for inhibiting T cell proliferation comprising contacting CD28 positive T cells with a soluble B7 fusion protein so as to bind CD28 on the CD28 positive T cells with the soluble B7 protein and thereby

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inhibiting T cell proliferation. According to the specification, page 12, "useful in the method of the invention is a B7Ig fusion protein that comprises a polypeptide corresponding to the extracellular domain of the B7 antigen and an immunoglobulin constant region that alters the solubility, affinity and/or valency (valency is herein defined as the number of binding sites available per molecule) of the B7 antigen." Administration of B7 antigen, e.g., as a soluble B7Ig fusion protein to react with CD28 positive T cells, will bind the CD28 receptor on the T cells and result in inhibition of the functional responses of T cells. Specification, page 21.

Under conditions where T cell interactions are occurring as a result of contact between T cells and B cells, binding of introduced B7 antigen in the form of a fusion protein that binds to CD28 receptor on CD28 positive T cells should interfere, i.e., inhibit, the T cell interactions with B cells. Likewise, administration of the CD28 antigen, or its fragments or derivatives, in vivo, for example in the form of a soluble CD28Ig fusion protein, will result in binding of the soluble CD28 Ig to B7 antigen, preventing the endogenous stimulation of CD28 receptor by B7 positive cells, such as activated B cells, and interfering with the interaction of B7 positive cells with T cells. *Id.*

In addition, the B7 fusion proteins may be used to regulate T cell proliferation. For example, the soluble CD28Ig and B7Ig fusion proteins may be used to block T cell proliferation in graft versus host (GVH) disease which accompanies allogenic bone marrow transplantation. Thus the B7 antigen in the form of B7Ig fusion protein, or in combination with immunosuppressants such as cyclosporin, may be used for blocking

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T cell proliferation in GVH disease. Specification page 26. In addition, B7Ig may be used to crosslink the CD28 receptor, for example by contacting T cells with immobilized B7Ig fusion protein, to assist in recovery of immune function after bone marrow transplantation by stimulating T cell proliferation. Specification page 27.

The fusion proteins may be useful to regulate granulocyte macrophage colony stimulating factor levels for the treatment of cancers, AIDS, and myelodysplasia. Ig. Moreover, the inhibition of anti-CD28 and anti B7mAbs on the cognate Th:B interaction also provides the basis for employing the CD28Ig and B7Ig fusion proteins to treat various autoimmune disorders associated with exaggerated B cell activation such as insulin-dependent diabetes mellitus, myasthenia gravis, rheumatoid arthritis and systemic lupus erythematosus (SLE). Specification, page 72. Methods for preparing B7Ig fusions proteins are described in the specification at pages 52-55.

#### Grounds of Rejection

Claims 79-94 stand rejected under 35 U.S.C. § 112, first paragraph, for lack of enablement.

Claims 79-94 stand rejected under 35 U.S.C. § 112, first paragraph and second paragraph for failing to define the invention in a manner as to enable any person skilled in the art to make and use the invention and for failing to point out and distinctly claim the invention.

We reverse both rejections for the reasons herein.

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### DISCUSSION

In reaching our decision in this appeal, we have given consideration to the appellants' specification and claims, to the applied references, and to the respective positions articulated by the appellants and the examiner.

Rather than reiterate the conflicting viewpoints advanced by the examiner and the appellants regarding the noted rejections, we make reference to the Examiner's Answer for the examiner's reasoning in support of the rejection, and to the appellants' Brief and Reply Brief for the appellants' arguments thereagainst. As a consequence of our review, we make the determinations which follow.

### Claim Interpretation

Our appellate reviewing court stated in Panduit Corp. v. Dennison Mfg. Co., 810 F.2d 1561, 1567-1568, 1 USPQ2d 1593, 1597 (Fed. Cir. 1987):

Analysis begins with a key legal question -- what is the invention claimed? Courts are required to view the claimed invention as a whole. 35 U.S.C. 103. Claim interpretation, in light of the specification, claim language, other claims and prosecution history, is a matter of law and will normally control the remainder of the decisional process. [Footnote omitted.]

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To that end, we also note that during ex parte prosecution, claims are to be given their broadest reasonable interpretation consistent with the description of the invention in the specification. In re Zletz, 893 F.2d 319, 321, 13 USPQ2d 1320, 1322 (Fed. Cir. 1989).

We interpret the term "B7" consistent with the specification pages 6 and 11 as that described in Freeman 1989, which is now referred to in the art as B7-1.

We interpret the term "B7 fusion protein" and "soluble" according to their ordinary definitions (a protein consisting of B7 fused to another protein, and water soluble, respectively) which is consistent with their specification usage.

We interpret the term "inhibit" in the claims, consistent with the specification page 64 and Figures 12 and 16, to include something less than complete inhibition or blocking of the CD28 receptor on T cells, such as binding or blocking a portion of CD28 on CD28 positive T cells with soluble B7 protein, such that some inhibition of T cell proliferation occurs.

35 U.S.C. §112, first paragraph

Claims 79-94 stand rejected under 35 U.S.C. §112, first paragraph, for lack of enablement. The Answer suggests that the claims contain subject matter which was not described in the specification in such a way to enable one of ordinary skill in the art to which it pertains to make and/or use the invention. Answer, page 2. The examiner's statement of rejection, however, appears to focus on lack of enablement as to the how

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to use the claimed invention. Thus, we will limit our decision to this aspect of the enablement rejection.

Although not explicitly stated in section 112, to be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without "undue experimentation." In re Vaeck, 947 F.2d 488, 495, 20 USPQ2d 1438, 1444 (Fed. Cir. 1991); In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404, (Fed. Cir. 1988); In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) (the first paragraph of section 112 requires that the scope of protection sought in a claim bear a reasonable correlation to the scope of enablement provided by the specification). "To be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without 'undue experimentation.'" [Emphasis added.] Genentech, Inc. v. Novo Nordisk, A/S, 108 F.3d 1361, 1365, 42 USPQ2d 1001, 1004 (Fed. Cir. 1997) (quoting In re Wright, 999 F.2d 1557, 1561, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993)). Conversely, the first paragraph of section 112 requires that the scope of protection sought in a claim bear a reasonable correlation to the scope of enablement provided by the specification. Nothing more than objective enablement is required, and therefore it is irrelevant whether this teaching is provided through broad terminology or illustrative examples. In re Marzocchi, 439 F.2d 220, 223, 169 USPQ 367, 369 (CCPA 1971).

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An analysis of whether the claims under appeal are supported by an enabling disclosure requires a determination of whether that disclosure contained sufficient information regarding the subject matter of the appealed claims as to enable one skilled in the pertinent art to make and use the claimed invention.

In order to establish a prima facie case of lack of enablement, the examiner has the initial burden to establish a reasonable basis to question the enablement provided for the claimed invention. See In re Wright, 999 F.2d 1557, 1561-62, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993) (examiner must provide a reasonable explanation as to why the scope of protection provided by a claim is not adequately enabled by the disclosure). See also In re Morehouse, 545 F2d 162, 192 USPQ 29 (CCPA 1976). The threshold step in resolving this issue is to determine whether the examiner has met his burden of proof by advancing acceptable reasoning inconsistent with enablement.

In support of the rejection for lack of enablement, the examiner argues (Answer, pages 2-3):

In vitro and animal model studies have not correlated well with in vivo clinical trials in patients. Since the therapeutic indices of immunosuppressive drugs such as adhesion-based biopharmaceutical drugs can be species- and model-dependent, it is not clear that reliance on the experimental observations of inhibiting cognate T:B interaction s [sic] with anti-CD28 antibodies and anti-B7 antibodies provides the basis for employing CD28Ig and B7 Ig fusion proteins (CD28 immunoglobulin fusion protein and B7 immunoglobulin fusion protein)... It is noted that B7Ig inhibited CD28-mediated adhesion in vitro to a lesser degree than the CD28-specific antibody 9.3 and that CD28Ig did not inhibit said in vitro adhesion (see page 64 of the instant specification). In addition, B7Ig in solution showed a modest enhancement of proliferation of T cells in vitro even though anti-CD28 antibody 9.3 was effective (page 65 of the instant

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specification). There is no objective evidence that CD28Ig was tested in this *in vitro* system or other experimental *in vitro* or *in vivo* systems that would be predictive of the therapeutic methods encompassed by the claims. There is insufficient objective evidence that accurately reflects the relative efficiency of the claimed methods to inhibit T cell proliferation or to prevent binding of CD28 receptor to B7 antigen, commensurate in scope with the therapeutic methods encompassed by the claimed invention.

As evidentiary support for lack of enablement the examiner relies on Kahan for establishing that no *in vitro* assay predicts or correlates with *in vivo* immuno-suppressive efficacy. Answer, page 3. Blazar is relied on as teaching that issues such as tissue distribution, half-life, affinity and avidity obtained with various CD28-B7-specific reagents might prove to be highly important in achieving graft vs. host disease (GVHD) protection. According to the examiner, Blazar discloses that anti-CD80 (B7-1) or anti-CD86 (B7-2) antibodies were ineffective in preventing T cell CD8-mediated GVHD lethality, that each antibody was partially effective in CD4-mediated GVHD and that the combination of anti-CD80 and anti-CD86 antibodies were effective in preventing GVHD lethality in murine experimental models. *Id.*

The examiner also relies on Perrin for the disclosure that, in contrast to the effective treatment of disease with CTLA-4 Ig; anti-CD80 (B7-1) attenuated the first clinical disease episode but not the relapse, anti-CD86 (B7-2) had no significant effect on the course of disease, and the combined treatment with anti-CD80 plus anti-CD86 resulted in the exacerbation of disease. *Id.*

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Finally, the examiner finds that Yi-qun indicates that "it is clear that inhibition of T cell response to soluble antigens will require the blocking of both B7-2 and B7-1 to be effective. More important it is unlikely that ongoing T cell response will be susceptible to inhibition by anti-B7 reagents, for example in autoimmune disease." *Id.*

For their part, appellants first argue that, "one is not required to enable any more than what is claimed." Brief, page 7.<sup>1</sup> Appellants further argue that Lenschow, of record, provides "in vivo data which show that blocking the CD28 receptor from binding the B7 antigen using only CD28 results in manipulation of the immune system. Lenschow conclude that blocking the interaction of co-stimulatory molecules such as CD28-B7 may provide a new approach to immunosuppression." Brief, page 9. Appellants argue that, on the basis of this publication, a 35 U.S.C. § 101 rejection was withdrawn by the examiner, however, the lack of enablement rejection was improperly maintained in view of Lenschow. Brief, pages 11-12.

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<sup>1</sup> We note, the "invention" referred to in the enablement requirement of section 112 is the claimed invention. Lindemann Maschinenfabrik GMBH v. American Hoist and Derrick Co., 730 F.2d 1452, 1463, 221 USPQ 481, 489 (Fed. Cir. 1984) (the question is whether the disclosure is sufficient to enable those skilled in the art to practice the claimed invention); Raytheon Co. v. Roper Corp., 724 F.2d 951, 956, 220 USPQ 592, 596 (Fed. Cir. 1983). That claims are interpreted in light of the specification does not mean that everything expressed in the specification must be read into all the claims. On the contrary, as was said in Environmental Designs, Ltd. v. Union Oil Co. of California, 713 F.2d 693, 699, 218 USPQ 865, 870-71 (Fed. Cir. 1983); The specification must be sufficiently explicit and complete to enable one skilled in the art to practice the invention, while a claim defines only that which the patentee regards as his invention. 35 U.S.C. §112. The claim, not the specification, measures the invention.

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Appellants argue that they have "provided *in vivo* data confirming the *in vitro* results using a homologous molecule, namely CTLA4Ig (see Applicants' response dated August 24, 1992 of parent application, namely, U.S. Serial No. 722,101). This *in vivo* data strengthens Applicants' *in vitro* data." Brief, page 14. Furthermore appellants argue that NIH has approved several protocols involving the use of CD28. *Id.* Appellants argue that this cuts against the Patent Office's argument that the art in this area is so unpredictable that *in vitro* data are not acceptable. Brief, page 15. Additionally, appellants argue that ample guidance is provided by applicants as to how to make the B7 fusion proteins and how to carry out the claimed methods. Brief, page 17.

Factors to be considered by the examiner in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman, [230 USPQ 546, 547 (Bd Pat App Int 1986)]. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims. In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988).

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In considering the enablement rejection before us for review, we find the following passage from PPG Indus., Inc. v. Guardian Indus. Corp., 75 F.3d 1558, 1564, 37 USPQ2d 1618, 1623 (Fed. Cir. 1996) to be instructive.

In unpredictable art areas, this court has refused to find broad generic claims enabled by specifications that demonstrate the enablement of only one or a few embodiments and do not demonstrate with reasonable specificity how to make and use other potential embodiments across the full scope of the claim. See, e.g., In re Goodman, 11 F.3d 1046, 1050-52, 29 USPQ2d 2010, 2013-15 (Fed. Cir. 1993); Amgen, Inc. v. Chugai Pharmaceutical Co., 927 F.2d. 1200, 1212-14, 18 USPQ2d 1016, 1026-28 (Fed. Cir. 1991); In re Vaeck, 947 F.2d at 496, 20 USPQ2d at 1445. Enablement is lacking in those cases, the court has explained, because the undescribed embodiments cannot be made, based on the disclosure in the specification, without undue experimentation. But the question of undue experimentation is a matter of degree. The fact that some experimentation is necessary does not preclude enablement; what is required is that the amount of experimentation "must not be unduly extensive." Atlas Powder Co. v. E.I. DuPont De Nemours & Co., 750 F.2d 1569, 1576, 224 USPQ 409, 413 (Fed. Cir. 1984).

The Patent and Trademark Office Board of Appeals summarized the point well when it stated:

The test is not merely quantitative, since a considerable amount of experimentation is permissible, if it is merely routine, or if the specification in question provides a reasonable amount of guidance with respect to the direction in which the experimentation should proceed to enable the determination of how to practice a desired embodiment of the invention claimed. Ex parte Jackson, 217 USPQ 804, 807 (1982).

In the present case, we find, on balance, the appellants' evidence in support of enablement to be more convincing and relevant than the examiner's evidence in support of the position of lack of enablement.

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First, the examiner provides a reference, Kahan, which generally suggests that there are no in vitro immune assays which predict or correlate with immunosuppressive efficacy. However, appellants present in vivo data in Lenschow, a publication more specific to the technology in question, which showed that CTLA4Ig bound to both murine and human B7 and inhibited primary xenogeneic mixed lymphocyte reactions in vitro. Lenschow, page 790, column 1. Moreover, CTLA4Ig in vivo treatment resulted in prolonged donor specific unresponsiveness to human pancreas islet xenografts. Lenschow used a xenogeneic transplant in vivo model and indicated that "one advantage of the xenogeneic transplant model is the availability of a MAb to human B7 that does not react with mouse B7... Thus, the role of human B7-bearing antigen-presenting cells (APCs) could be directly examined." Lenschow, page 790, column 3. Therefore, it would reasonably appear that appellants have provided in vivo experimental evidence conducted in a relevant, art accepted model to support enablement of the pending claims. We also agree with appellants that the examiner has provided "no reason to believe that the use of B7 and CD28 antigens would be unpredictable in view of the successful use of homologous molecules, e.g., CTLA4Ig, in vivo." Brief, page 14. In our view, the data and discussion in Lenschow is more relevant to the enablement issue before us, than the general immunosuppression publication cited by the examiner, Kahan.

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Blazar is cited by the examiner for the proposition that the relevant art is unpredictable and that any conclusion regarding efficacy of the CD28/B7 blockade on altering the in vivo immune response should be interpreted in light of the type of reagent infused.<sup>2</sup> Answer, page 3. Blazar discloses that antiCD80 (B7-1) or anti-CD86 (B7-2) antibodies were ineffective in preventing T cell CD8-mediated GVHD lethality. The claimed invention, however, is directed to inhibiting T cell proliferation comprising contacting CD28 positive T cells with a soluble B7 fusion protein so as to bind CD28 on the CD28 positive T cells with the soluble B7 protein and thereby inhibiting T cell proliferation. The examiner has not explained the relevance of Blazar's prevention of T cell CD8-mediated GVHD lethality to the claimed inhibition of T cell proliferation by binding CD28 on the CD28 positive T cells with the soluble B7 fusion protein, in support of the position of lack of enablement of the claimed invention.

The appellants argue that the in vivo data in Lenschow describing CTLA4Ig, a molecule homologous to CD28 and which binds to both human and murine B7, supports enablement of the claimed invention. This would appear to be the same CTLA4Ig which was described in Blazar as reducing the GVHD capacity of donor T cells infused into fully allogenic recipients in vivo (Blazar, page 3250, column 2). Blazar

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<sup>2</sup> Appellants' invention relates to B7 (now referred to in the art as B7-1) fusion protein.

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also found that anti-CD80 (B7-1) and anti-CD86 (B7-2) antibodies<sup>3</sup> were partially effective in CD4+ mediated GVHD and that the combination of anti-CD80 and anti-CD86 antibodies were effective in preventing GVHD lethality in murine experimental models. Thus, arguably, Blazar would also lend some support to appellants' position that contacting CD28 positive T cells with a soluble B7 fusion protein homolog inhibits some degree of T cell proliferation.

Perrin, cited by the examiner, suggests that anti-CD80 (B7-1) attenuated the first clinical disease episode of experimental allergic myeloencephalitis but not the relapses and that CTLA4-Ig treatment resulted in attenuated disease, chiefly affecting subsequent relapses. Perrin, page 21, column 1. While noting a difference in anti-CD80 (B7-1) and CTLA4-Ig activity, Perrin would also reasonably support appellants' position that contacting CD28 positive T cells with a soluble B7 fusion protein homolog inhibits some degree of T cell proliferation.

Similarly, Yi-qun, Figure 1, evidences that anti-B7-1 provides some level of T-cell proliferation inhibition. Yi-qun describes that CTLA4-Ig or anti-CD28 Fab inhibits antigen specific T cell activation to the same extent as a combination of anti-B7-1 and anti-B7-2 mAbs (Figure 3).

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<sup>3</sup> CD80 (B7-1) and CD86 (B7-2) bind to CD28 and CTLA-4 counter-receptors on T cells. Blazar, page 3250, column 2.

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While we find our decision in this case has been made difficult by the failure of appellants to specifically address the references which the examiner has cited in support of lack of enablement, we find merit in appellants' position that Lenschow is supportive of enablement of the pending claims. The rejection of the claims for lack of enablement is reversed.

35 U.S.C. § 112, first and second paragraphs

Claims 79-94 stand rejected under 35 U.S.C. § 112, first paragraph and second paragraph for failing to define the invention in a manner as to enable any person skilled in the art to make and use the invention and for failing to point out and distinctly claim the invention.

As set forth in Amgen Inc. v. Chugai Pharmaceutical Co., Ltd., 927 F.2d 1200, 1217, 18 USPQ2d 1016, 1030 (Fed. Cir. 1991):

The statute requires that "[t]he specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention." A decision as to whether a claim is invalid under this provision requires a determination whether those skilled in the art would understand what is claimed. See Shatterproof Glass Corp. v. Libbey-Owens Ford Co., 758 F.2d 613, 624, 225 USPQ 634, 641 (Fed. Cir. 1985) (Claims must "reasonably apprise those skilled in the art" as to their scope and be "as precise as the subject matter permits.").

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Furthermore, claim language must be analyzed "not in a vacuum, but always in light of the teachings of the prior art and of the particular application disclosure as it would be interpreted by one possessing the ordinary skill in the pertinent art." In re Moore, 439 F.2d 1232, 1235, 169 USPQ 236, 238 (CCPA 1971).

The examiner argues that it is unclear what is meant by "B7" and by the phrase "containing amino acid residues from about position 1 to about position 215 of the amino acid sequence corresponding to the extracellular domain of B7 antigen because their characteristics are ambiguous and not defined." Answer, page 5. The examiner suggests that "while the name itself may have some notion of the activity of the protein, there is nothing in the claims which distinctly claims the protein and variants thereof. Others in the field may isolate the same protein and give such an entirely different name. Also B7 can refer to a number of distinct proteins expressed on various tissues and in various animal species." The examiner continues, "Claiming biochemical molecules by a particular name given to the protein by various workers in the field fails to distinctly claim what that protein is and what the compounds are made up of. This language is vague and indefinite since it can encompass many different proteins and is not apparent which particular antigen is being referred to." Answer, page 5.

As discussed herein, we have interpreted the term "B7" consistent with the prosecution history and specification pages 6 and 11 as that described in Freeman 1989, which is now referred to in the art as B7-1. We find no ambiguity in its meaning, as defined in the specification.

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The appellants have also countered the examiner's position, suggesting that the claim language is not indefinite as "[a]pplicants have provided the entire nucleotide sequence for one B7 protein and described the functions which other members of the class of proteins provided by the invention would have to have." Brief, page 18. The appellants argue that art searches in the field establish that "B7" is understood by those of ordinary skill in the art to be the protein having the characteristics of the protein as claimed. We agree.

Appellants argue that despite the fact that they do not disclose every known B7 molecule, the identification of other species in the class would not entail undue experimentation because Applicants' disclosure outlines a number of different assays for the identification of B7 molecules as claimed. See specification pages 43, 61 and 66. Brief, pages 18-19. We also agree that the specification has provided reasonable guidance to one of ordinary skill in the art to identify other species of B7 protein in the class without undue experimentation. The rejection of claims 79-94 under 35 U.S.C. § 112, first and second paragraphs are reversed.

### CONCLUSION

In view of the above, the rejections of claims 79-94 under 35 U.S.C. § 112, first paragraph and claims 79-94 under 35 U.S.C. § 112, first and second paragraphs, are reversed.

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No time period for taking any subsequent action in connection with this appeal  
may be extended under 37 CFR § 1.136(a).

REVERSED

*William F. Smith*  
WILLIAM F. SMITH  
Administrative Patent Judge

*Demetra J. Mills*  
DEMETRA J. MILLS  
Administrative Patent Judge

*Eric Grimes*  
ERIC GRIMES  
Administrative Patent Judge

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